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The co-occurrence between depressive symptoms and paranoid ideation: a population-based longitudinal study

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Abstract

Background: The aim of this study was to examine longitudinally in the general population (a) whether depressive symptoms co-occur with paranoid ideation from late adolescence to middle age (b) whether depressive subsymptoms are differently linked with paranoid ideation (c) whether depressive symptoms are associated with state-level or trait-level paranoid ideation.

Methods: Altogether 2109 subjects of the Young Finns study completed the Paranoid Ideation Scale of the Symptom Checklist-90 Revised and a modified version of the Beck Depression Inventory in 1992, 1997, 2001, 2007, and 2012, and the Beck Depression Inventory-II in 2007, 2011, and 2012.

Results: Higher self-rated depressive symptoms were associated with the course of more severe paranoid ideation over age, especially in late adolescence and early adulthood. Regarding depressive subsymptoms, the associations of negative attitude and performance difficulties with paranoid ideation were evident over age, whereas the influence of somatic symptoms (such as changes in sleep and appetite) was not significant until after early adulthood. Additionally, depressive symptoms were more evidently associated with the development of trait- than state-level paranoid ideation.

Limitations: Our study mostly captured mild depressive and paranoid symptoms. The results cannot be directly generalized to clinical populations.

Conclusions: Depressive symptoms were associated with the course of paranoid ideation from late adolescence to middle age. Patients with paranoid ideation might merit from evaluation of potential depressive symptoms, especially in late adolescence and early adulthood. Among patients with co-occurring depressive symptoms and paranoid ideation, there may be a substantial need for neurocognitive rehabilitation and community-based treatments.

Keywords

depression; co-occurrence; longitudinal; paranoia; population-based

1. Introduction

It has been widely recognized, that there is excessive comorbidity between psychiatric disorders (Valderas, 2009; Widiger & Samuel, 2005). The co-occurrence of psychiatric symptoms is estimated to be much more common than has been obtained at diagnostic level, so that patients with only a single disorder may in reality represent an atypical minority (Brown & Barlow, 2005; Hyman, 2010). Recently, it has been emphasized that comorbidity should be utilized as a tool for recognizing the full range of targets for treatment more widely than previously (Dell'Osso & Pini, 2012). Along with this, diagnostic classifications have strived for increasing comorbidity rates by reducing the number of mutually exclusive diagnoses (First, 2005). Hence, there is a growing demand to understand the interaction and course of co-occurring symptoms.

In the present study, we examine the comorbidity between depressive symptoms and paranoid ideation, which has provoked substantial interest over decades. Paranoia is defined as an unjustified distrust and suspiciousness towards others so that their motives are interpreted as malevolent (APA, 2013). Mild paranoia refers to feelings of vulnerability in interpersonal relationships, whereas most severe paranoid symptoms can be diagnosed as paranoid personality disorder or even as psychotic-level persecutory delusions (Freeman et al., 2011; Freeman & Garety, 2014). To date, there is a great body of evidence that depressive symptoms correlate with paranoid ideation both at clinical and subclinical levels (e.g. Freeman et al., 2008, 2010, 2012; Grant et al., 2005; Kool et al., 2000; Martin & Penn, 2001). Above 25% of depressed patients have clinical paranoid symptoms (Kool et al., 2000; Ramklint & Ekselius, 2003). However, long-term follow-up studies examining the co-occurrence

and interaction of depressive symptoms and paranoid ideation in non-clinical adults are missing. Hence, there are still a range of open questions.

The first question is whether depressive symptoms are associated with paranoid ideation longitudinally over age. Several previous findings have suggested that depressive symptoms predict the onset and persistence of paranoid symptoms within adulthood (e.g. Fowler et al., 2011; Freeman et al., 2012; Vorontsova et al., 2013). Some studies, however, have indicated that the predictive pathways from depressive symptoms to paranoid ideation are relatively weak (Moritz et al., 2017) or even non-significant (Drake et al., 2004). By now, the follow-up periods have been at most 24 months. Hence, theoretic models of paranoia have suggested a predisposing and maintaining role for specific depressive symptoms, but highlighted the need for further evidence from longitudinal studies (Freeman & Garety, 2014).

A second question is whether different depressive subsymptoms, such as negative attitude, performance difficulties, and somatic symptoms, are differently associated with paranoid ideation. It has been recommended to examine symptoms separately (Fried & Nesse, 2015), because patients with depressive disorders may have varying symptom profiles (APA, 2013) and different symptoms may be linked with different comorbidity patterns (Lux & Kendler, 2010). Previously, negative attitude is found to be associated with paranoid ideation in clinical populations (Bentall et al., 2009; Corcoran, 2006; Vorontsova et al., 2013). The association between negative attitude and paranoid ideation, however, might be mediated via performance difficulties. This is because performance difficulties, such as weaker social and executive functioning, have been found to correlate with negative attitude (Snyder et al., 2013) and also with higher paranoia (Freeman et al., 2011; Bentall et al., 2009; Vorontsova et al., 2013). Regarding somatic complaints, previous studies have reported that insomnia, weight change, and sensory disturbances are associated with paranoid symptoms (Freeman et al., 2011, 2012). This association might be indirect via other risk factors such as current

mood and performance difficulties (Freeman et al., 2002). However, there has been no study simultaneously investigating the associations of all depressive subsymptoms with paranoid ideation.

Recently, there has been a call for studies delineating between state- and trait-level paranoia (Corcoran et al., 2006). Hence, the third question is whether depressive symptoms are associated with state- or trait-level paranoia, i.e. whether paranoid ideas appear and disappear along depressive episodes or whether they persist also after the stabilization of acute depressive state. By now, there is evidence for an association of depressive symptoms with state paranoia: experimental studies have suggested that depressive mood increases the risk for paranoid ideas during the following days (Freeman et al., 2008; Thewissen et al., 2011) and improvement of depressive symptoms has been shown to predict decrease in symptoms of paranoid personality disorder over the following weeks (Fava et al., 2002). Regarding a trait paranoia, based on clinical observations only chronic depression has been suggested to be linked with trait-level increase in paranoia (Hirschfeld, 1999). However, studies with long follow-ups would be needed to capture potential trait-level changes in paranoid ideation related to depressive symptoms.

Currently, the comorbidity between depression and paranoia constitutes a considerable strain for health-care system. It has been demonstrated that among patients with clinical depression, comorbid paranoid symptoms are associated with poorer treatment outcome (Joyce et al., 2007; Mrazek et al., 2014; Skodol et al., 2011). Correspondingly, the current psychotherapeutic interventions for paranoid ideation appear to be ineffective (Dixon-Gordon et al., 2011; Karterud et al., 2004; Schneider & Klauer, 2001), which in many cases is supposed to result from co-occurring depression (Bockian, 2006). Thus, revealing the unknown aspects of this comorbidity, i.e. which depressive symptoms are associated with paranoid ideation and whether the associations persist over age, will have implications for tailoring more effective interventions especially for treatment-resistant patient populations.

The aim of our study was to investigate longitudinally in a non-clinical population (a)

whether there is a co-occurrence between depressive symptoms and paranoid ideation from adolescence to middle age (b) whether depressive subsymptoms are differently linked with paranoid ideation (c) whether depressive symptoms are associated with state- or trait-level course of paranoid ideation, i.e. whether paranoid ideation appears and disappears along depressive episodes. Our data with a 20-year prospective follow-up and several measurement points provides a unique possibility for investigating how these associations are shaped over the lifespan.

2. Methods

2.1 Participants

We used data from the prospective Young Finns Study (YFS). Participants were selected from the population register of the Social Insurance Institution. The original sample included 3596 participants that were selected haphazardly from six age cohorts (born in 1962, 1965, 1968, 1971, 1974, and 1977). The baseline measurement was in 1980, and the participants have been followed since then so that the most recent measurement time was in 2012 (participants were aged 35–50). The study was carried out in accordance with the Declaration of Helsinki, and the study design was approved by Finnish Advisory Board on Research Integrity. Before participation, all the participants or their parents provided informed consent after the nature of the procedures had been fully explained. The design of the YFS is described more exactly elsewhere (Raitakari et al., 2002).

For this study, depressive symptoms were measured with a modified version of the Beck Depression Inventory-II (BDI-II) in 1992, 1997, 2001, 2007, and 2012 and with the BDI-II in 2007, 2011, and 2012. Paranoid ideation was measured in 1992, 1997, 2001, 2007, and 2012, socioeconomic factors in 2001 and 2007, and parental socioeconomic factors in 1980. We included in the analyses all participants with full data (i.e. no missing values) on paranoid ideation, depressive symptoms, and their

own and parental socioeconomic status in at least one of the measurement times (full data about paranoid ideation in 1992, 1997, 2001, 2007, or 2012; full data about socioeconomic status in 2001 or 2007 etc.). The final data consisted of 2109 participants. The numbers of person-observations are shown in Supplemental Table 1.

2.2. Measures

Paranoid ideation was measured with the Paranoid Ideation Scale of the Symptom Checklist-90 Revised (SCL-90R; Derogatis, 1986). It consists of 6 self-rating items that are answered with a 5-point Likert-Scale (1=totally disagree, 5=totally agree). Earlier studies have found high reliability and predictive validity for the scale (e.g. Olsen et al., 2004; Schmitz et al., 2000). Based on previous recommendations (e.g. Freeman & Garety, 2014), we treated paranoia as continuous dimension and calculated a sum score of the items for each measurement time.

Depressive symptoms were measured with the Beck Depression Inventory-II (BDI-II; Beck, Steer & Brown, 1996) and with a modified version of BDI (mBDI). The BDI-II consists of 21 items with four answer options under each item, ranging from not present (0) to severe (3). It is a widely used and internationally validated measure of depression (e.g. Nuevo et al., 2009; Storch et al., 2004). We computed a four-class variable indicating the severity of depression: no depression (scores 0–13), mild (14–19), moderate (20–28), or severe depression (29–63) (Beck et al., 1996).

The modified BDI (mBDI) includes the second mildest statements of the BDI, and the statements were answered with a 5-point Likert-scale (1=totally disagree, 5=totally agree). Thus, it captures mild depressive symptoms more sensitively than the original BDI and is an especially valuable measure when investigating depressive symptoms in the general population (e.g. Katainen et al., 1999; Nurmi et al., 1995; Rosenström et al., 2012). The mBDI consists of three factors: negative attitude, performance difficulties, and somatic complaints. The mBDI has been used in earlier studies, too (e.g.

Oikonen et al., 2014), and a detailed description of the measure has been published previously (Elovainio et al., 2005). In the current study, we computed a total score for depressive symptoms and scores for the depressive subsymptoms at each measurement time. The internal consistencies for the scales of depressive symptoms and paranoid ideation are shown in Supplemental Table 2. For each included participant, the scores for depressive symptoms and paranoid ideation were calculated for all the measurement points with no missing values in the concerned scale.

Participants' and their parents' socioeconomic factors included occupational status and the number of educational years. The number of educational years was standardized with the mean of 0 and standard deviation of 1. Occupational status was measured according to the year 1979 (parents) or 2001 (participants) classification of the Center of Statistics in Finland and categorized as manual, lower grade non-manual, or upper grade non-manual. All the socioeconomic variables were added to the analyses as separate variables.

2.3. Statistical analyses

Statistical analyses were conducted with Stata SE version 13.0. Attrition was examined by comparing our study variables of the included (n=2109) and excluded (n=1487) participants. We examined the longitudinal associations between depressive symptoms and paranoid ideation with multilevel models (MLMs) using maximum likelihood estimation. MLMs can estimate simultaneously fixed effects (classic regression coefficients) and random effects that refer to between-individual variance in intercept and slopes. Coefficients of determination were based on Cox and Snell's generalized R squared (Cox & Snell, 1989).

Model 1 was an unconditional growth model for paranoid ideation with intercept, age, age-squared, gender, and socioeconomic factors as fixed effects, and intercept and age as random

effects. Age referred to participant's age at each measurement point, ranging 15 (the age of the youngest cohort in 1992) to 50 (the age of the oldest cohort in 2012). Age was centered at age 15. In model 2, we examined the effect of depressive symptoms (measured with the mBDI) on paranoid ideation. We added depressive symptoms and their age-interactions to fixed effects. Next in models 3, we examined the associations of depressive subsymptoms with paranoid ideation. We added separately each subsymptom score and its age-interaction to fixed effects. In model 4, we added simultaneously all the subsymptom scores and their age-interactions to fixed effects. In all the models, except for the unconditional model, depressive symptoms were added also to random effects.

The associations of depressive symptoms with trait- and state-level paranoia were investigated by comparing whether adding depressive symptoms to the unconditional model reduced the variance of intercept (i.e. between-individual variation of paranoid ideation over the follow-up) or the residual variance of paranoia (i.e. within-individual variation between measurement times). That is, whether depressive symptoms explained between-individual variation in paranoia (trait paranoia) rather than within-individual variation in paranoia (state paranoia).

As additional analysis (models 5), our data also enabled to examine the association of changes in depressive symptoms with changes in paranoid ideation. We used year 1992 scores as baseline levels for depressive symptoms and paranoid ideation. For year 1997, 2001, 2007, and 2012 scores, we calculated their difference with the preceding measurement point and used them as indicators for the change between measurement times. We added intercept, age, age-squared, change in depressive symptoms and its age-interaction, baseline paranoid ideation, baseline depressive symptoms, gender, and socioeconomic factors to fixed effects. Intercept was treated also as random effect.

Finally, we examined the associations of the BDI-II depressive symptoms with paranoid ideation using regression analyses. We predicted paranoid ideation in 2012 by severity of depression in

2007, 2011, and 2012. The models were adjusted for the level of paranoid ideation in 2007, age, gender, and socioeconomic factors.

3. Results

Descriptive statistics are shown in Table 1. Attrition analyses revealed that women were more likely to participate than men (65.4% vs. 51.6%, $p<.001$). Participants had a lower total score in depressive symptoms than non-participants assessed both with mBDI (43.91 vs. 46.65, $p<.001$) and BDI-II (5.09 vs. 6.22, $p<.001$). They also had a lower score in negative attitude (16.61 vs. 17.76, $p<.001$), performance difficulties (14.10 vs. 15.19, $p<.001$), and somatic complaints (12.76 vs. 13.14, $p<.01$). Participants had also lower paranoid ideation than non-participants (13.60 vs. 14.78, $p<.001$), more educational years (15.38 vs. 14.08, $p<.001$), were less likely to be manual or lower non-manual workers (52.5% vs. 69.4%, $p<.001$), and their parents had more educational years (10.78 vs. 10.52, $p<.05$).

[Table 1]

The associations between depressive symptoms (the mBDI) and paranoid ideation are shown in Table 2. The fixed effects indicated that higher total score of depressive symptoms was associated with the development of higher paranoid ideation. This association was evident over the follow-up even though it slightly decreased over age (Figure 1a), as indicated by the age-interaction of depressive symptoms. When added separately to the model, higher negative attitude, higher performance difficulties, and higher somatic complaints were associated with more severe paranoid ideation. Instead, when adding them simultaneously, the main effect of somatic complaints was

nonsignificant. However, the age-interaction of somatic complaints revealed that the effect of somatic complaints on paranoid ideation became significant over age (Figure 1b).

[Table 2]

[Figure 1]

Regarding the development of state vs. trait paranoia, our results indicated that after adding depressive symptoms to the unconditional model, the decrease was roughly greater in variance of intercept (between-individual variation of paranoia) than in residual variance (within-individual variation of paranoia). That is, depressive symptoms appeared to be associated more strongly with trait- than state-like paranoia.

As additional analyses, we reanalyzed models 1–4 so that the sum score of paranoid ideation was confined to truly paranoid items (excluding items ”I have ideas that others do not share” and ”Others do not appreciate my achievements sufficiently”). All the results remained, even slightly strengthened. Additionally, by using multilevel models corresponding to the models 1–4, we examined the effects of such single depressive symptoms, that have been proposed to have an essential role for the development of paranoid ideation (Freeman et al., 2002; Freeman & Garety, 2014). The results revealed that all these very symptoms, i.e. more severe sleep disturbances (fixed effect: $\beta=.16$, $p<.001$), higher social disinterest ($\beta=.20$, $p<.001$), more severe impairment in initiative ($\beta=.19$, $p<.001$), higher rumination (thinking about one’s mistakes) ($\beta=.23$, $p<.001$), higher depressive mood ($\beta=.26$, $p<.001$), and higher worry ($\beta=.32$, $p<.001$) were associated with the development of higher paranoid ideation.

Changes in depressive symptoms were positively associated with changes in paranoid ideation (Table 3), i.e. increase in depressive symptoms was associated with increase in the level of

paranoid ideation. This association, however, became weaker over age, that was revealed by the age-interaction of change in depressive symptoms (Figure 2). Thus, over age, the level of paranoid ideation seemed to become more stable in relation to changes in depressive symptoms. The results remained, when confining the scores of paranoid ideation to truly paranoid items.

[Table 3]

[Figure 2]

Regarding depressive symptoms measured with the BDI-II, the severity of depression in 2007 was not significantly associated with the development of paranoid ideation in 2012 (Table 4). Instead, more severe level of depression in 2011 and 2012 was associated with higher paranoid ideation in 2012. Participants with severe depression had higher paranoid ideation than participants with mild ($p<.05$) or moderate depression ($p<.01$). The results were replicated, when confining the scores of paranoid ideation to truly paranoid items.

[Table 4]

As supplementary analyses, we examined the causal relationships between depressive symptoms (mBDI) and paranoid ideation in 1992, 1997, 2001, 2007, and 2012 using cross-lagged panel design for longitudinal data. Three models were estimated: (1) a model with only stability coefficients (predictive paths between depressive symptom scores at different timepoints and between paranoid ideation scores at different timepoints) and covariances between depressive symptoms and paranoid ideation at each timepoint (2) a model including also cross-lagged predictive paths from depressive

symptoms at each timepoint to paranoid ideation at the following timepoint, and vice versa (constrained to be equal in both directions) (3) a model with the cross-lagged predictive paths freely estimated. The goodness-of-fit statistics of the models are shown in Supplemental Table 3. Models 2 and 3 had significantly better fit than model 1, suggesting that there were predictive relationships between depressive symptoms and paranoid ideation. Model 2 had good fit, but model 3 had significantly better fit than model 2. Model 3 revealed that there were significant positive predictive associations in both directions, but the predictive coefficients were stronger from depressive symptoms to paranoid ideation than vice versa. Moreover, the results remained, when the sum scores of paranoid ideation were confined to truly paranoid items.

4. Discussion

Our study is the first to investigate the comorbidity between depressive symptoms and paranoid ideation in the general population longitudinally from late adolescence to middle age. Our results revealed that depressive symptoms were associated with the course of more severe paranoid ideation, especially in late adolescence and early adulthood. Regarding depressive subsymptoms, the influences of negative attitude and performance difficulties on paranoid ideation were evident over age, whereas the influence of somatic symptoms was not significant until after early adulthood. With regard to single depressive symptoms, we found that higher depressive mood, higher worry, higher rumination (thinking about one's mistakes), more severe sleep disturbances, higher social disinterest, and more severe impairment in initiative were associated with the development of higher paranoid ideation. This is in line with previous postulations (Freeman et al., 2002; Freeman & Garety, 2014). Additionally, depressive symptoms were associated more strongly with the development of trait- than state-level paranoid ideation, indicating that paranoid ideation reflected rather a dispositional trait than a state that

might appear and disappear along depressive episodes. These associations sustained after controlling for age, gender, socioeconomic factors, and individual-level variation in the influences of depressive symptoms. Furthermore, the results remained, when confining the scores of paranoid ideation to truly paranoid items.

Negative attitude, which reflects the symptoms that are regarded as the core symptoms of depressive disorders (APA, 2013), was associated with paranoid ideation even when controlling for performance difficulties, that are characteristic also for a range of other psychiatric disorders (Elliott et al., 2003; Moritz et al., 2002). Thus, our results indicate that the co-occurrence between depressive symptoms and paranoid ideation may not only reflect a general liability to psychiatric disorders, but depressive and paranoid ideas may include partly overlapping cognitive-emotional processes. For example, previous studies have suggested that both depression and paranoid ideation correlate with beliefs about others' negative evaluation toward the self (Martin & Penn, 2001) and expectations that pleasant events are unlikely to happen to the self (Corcoran et al., 2006). Our study demonstrated that the association of negative attitude with paranoid ideation remains highly similar from late adolescence to middle age.

Our findings also revealed that performance difficulties, i.e. depression-related impairments in executive functioning and lowered social activity, were linked with the development of higher paranoid ideation. This is in line with an environmental feedback hypothesis (Morse & Lynch, 2004) postulating that when staying away from interpersonal situations, an individual has fewer possibilities to encounter contradictory evidence for paranoid beliefs. Furthermore, impaired executive functioning is likely to extend the persistence of paranoid symptoms by lowering the ability to control arbitrary interpretations for others' purposes (Freeman et al., 2002*).*

When controlling for negative attitude and performance difficulties, the main effect of somatic complaints on paranoid ideation became nonsignificant. This is in line with the recent

developmental models of paranoia postulating that specific somatic symptoms are associated with paranoid ideation indirectly by predisposing to biases in affective and cognitive processing (Freeman & Garety, 2014). Our study also confirmed the previously found association of sleep disturbances with higher paranoid ideation (Freeman et al., 2010). Insomnia may contribute to the development of paranoid symptoms by increasing the risk for transient deficits in cognitive control (Freeman et al., 2010; Freeman & Garety, 2014). Over age, however, the influence of somatic complaints became significant, indicating that there is a more direct association between somatic complaints and paranoid ideation after early adulthood. This might refer to such cases in which somatic complaints partly form the content of paranoid ideation, so that somatic symptoms are interpreted as markers of damage caused by others (APA, 2013). The association of somatic symptoms with paranoia may become stronger over age also because somatic symptoms are especially characteristic for late-onset depression (Fiske et al., 2009) and because biased interpretations for benign bodily sensations are more common in older ages (Suvisaari et al., 2009).

A concern has been expressed that recording state-level comorbidities of psychiatric symptoms may, in some cases, result in short-term diagnostic instability (Widiger & Samuel, 2005). Regarding the co-occurrence between depression and paranoia, however, this risk seems not to be justifiable since depressive symptoms appeared to be more strongly associated with trait- than state-level paranoid ideation. Recently, there has been a growing demand for investigating which factors might contribute to the development of late-onset personality disorder traits (Oltmanns & Balsis, 2011; van Alphen et al., 2015). In our study, the association of changes in depressive symptoms with changes in paranoid ideation became weaker over age. This finding tentatively suggests that late-onset depressive symptoms may not play a significant role in the appearance of paranoid traits.

Our results also suggested that the relationship of depressive symptoms with paranoid ideation is especially strong at severe levels of depression. It may be that suicidality, which is

characteristic especially for severe depression (APA, 2013), intensifies the link between depression and paranoia, because both suicidal and paranoid ideation include feelings of guilt, personal inadequacy, and deserved punishment (Freeman et al., 2011). Actually, there have been contradictory views whether there even exists severe depression without any paranoid ideation (e.g. Keller et al., 2007; Schatzberg et al., 1992).

Our results also suggested that the predictive associations from paranoid ideation to depressive symptoms are weaker than in the opposite direction, but still significant. This is contrary to Fowler's et al. (2011) findings. Overall, there is previous evidence that especially psychotic-level delusional ideation predicts the development of depressive symptomatology (e.g. Birchwood et al., 2000, 2005; Iqbal et al., 2000; Sullivan et al., 2014). It has been demonstrated that higher insight of delusional beliefs and a more negative appraisal of one's delusion-related diagnosis have crucial roles for the development of later depression (Birchwood et al., 2005; Iqbal et al., 2000). For example, a more negative appraisal may lead to feelings of humiliation due to receiving a delusion-related diagnosis, impaired self-confidence, experience of loss of one's occupational roles, adoption of a lower social status, interpersonal avoidance, and eventually to depression (Birchwood et al., 2005; Iqbal et al., 2000; Sullivan et al., 2014). Hence, the predictive pathway from paranoid ideation to depressive symptoms may be stronger in clinical samples including more severe and diagnostically significant levels of paranoid ideation than in our non-clinical sample.

This study had some limitations. First, as usual in studies with long follow-ups, there was significant attrition. Non-participants had on average more severe depressive symptoms and higher paranoid ideation. It is a common finding that participants with high paranoia or other psychiatric symptoms are more likely to drop out during follow-up (Graaf et al., 2000; Triebwasser et al., 2013). Hence, our study may have mostly captured milder symptoms so that the results cannot be directly generalized to clinical populations. It may also be possible that the co-occurrence between depression

and paranoia had been more evident in clinical populations. However, our primary goal was to investigate mild subclinical symptoms in a non-clinical population to acquire tools for developing early preventive treatments and improving the diagnostic accuracy, as has been recommended earlier (Freeman & Garety, 2014). For this reason, we also used a modified version of the BDI over the 20-year follow-up so that we could capture sensitively the variation at mild levels of depressive symptoms.

Secondly, the results regarding somatic symptoms must be interpreted with caution, because Cronbach's alpha for the somatic symptoms scale was low in year 1992 measurement point. Reliability analyses revealed that deleting any single item would not have improved the internal consistency of the scale significantly. Further examination revealed that all items of the scale had correlations of 0.5 or higher with the overall construct of somatic complaints in 1992. This indicates adequate internal consistency for the scale despite the comparatively low alpha for the scale in 1992. The low alpha of this subscale is likely explained by the fact that depressive disorders include highly heterogeneous profiles of somatic symptoms (Fried & Nesse, 2015), so that distinct somatic symptoms may even include dysfunctions in opposite directions (Kapfhammer, 2006), for example, decreased need for sleep and increased appetite.

Our study had also several strengths. We used prospective data with a long follow-up and several measurement points, which provided unique possibilities to investigate the co-occurrence between depressive symptoms and paranoid ideation from late adolescence to middle age. Furthermore, we could take into account many potential sociodemographic confounding factors and individual-level variation in the associations. We also had a large population-based sample which, despite some selective attrition, is still likely to represent the general population with regard to most characteristics.

This study has several implications for the clinical work of mental health services. Patients with paranoid ideas are likely to merit from evaluation of potential depressive symptoms, especially in late adolescence and early adulthood, when the link between depressive symptoms and

paranoid ideation is particularly strong. When tailoring interventions for middle-aged patients, it is necessary to screen somatic complaints because their potential role for the development of paranoid ideation may otherwise remain unrecognized. Furthermore, among patients with co-occurring depressive and paranoid symptoms, there may be a substantial need for neurocognitive rehabilitation as well as community-based treatments to enhance interpersonal activities. In conclusion, our results are in accordance with recent reviews postulating that the treatment of specific depressive symptoms could reduce the predisposing and maintaining factors of paranoid ideation (Freeman & Garety, 2014; Lake, 2008).

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Table 1. Means, standard deviations (SD), frequencies, and ranges of the variables under study.

	Mean / Frequency (%)	SD	Range
Age in 1992	22.50	5.01	15–50
Gender			
Female	1199 (56.9)		
Male	910 (43.1)		
Educational years	15.38	3.42	8–32
Parental educational years	10.78	3.59	1–28
Occupational status			
Manual	831 (39.4)		
Lower grade non-manual	385 (18.3)		
Upper grade non-manual	1001 (47.5)		
Parental occupational status			
Manual	831 (39.4)		
Lower grade non-manual	912 (43.2)		
Upper grade non-manual	366 (17.4)		
Paranoid ideation[a]	13.60	3.26	6-27.8
The MBDI[a]			
Total score	43.91	11.13	21–88.
Negative attitude	16.61	4.49	8–35
Performance difficulties	14.10	4.39	7–33
Somatic complaints	12.76	3.38	6–24
The BDI-II			
Total score[a]	5.09	5.74	0–43
Severity of depression[b]			
Mild	201 (9.5)		
Moderate	88 (4.2)		
Severe	36 (1.7)		

[a] The mean over the follow-up. [b] The frequency of participants having the certain level of depression in at least one measurement time.

Table 2. Estimates and standard errors (SE) of fixed and random effects when predicting standardized scores of paranoid ideation by depressive symptoms (measured with the mBDI) and age.

	Model 1 (R^2=.14) Estimate (SE)	Model 2 (R^2=.55) Estimate (SE)	Model 3a (R^2=.50) Estimate (SE)	Model 3b (R^2=.49) Estimate (SE)	Model 3c (R^2=.33) Estimate (SE)	Model 4 (R^2=.56) Estimate (SE)
Fixed effects						
Intercept	0.93 (0.11)***	0.23 (0.04)***	0.29 (0.04)***	0.24 (0.04)***	0.33 (0.05)***	0.59 (0.10)***
Total depressive symptoms		0.57 (0.03)***				
Negative attitude			0.47 (0.02)***			0.32 (0.05)***
Performance difficulties				0.47 (0.02)***		0.29 (0.06)***
Somatic complaints					0.26 (0.02)***	-0.072 (0.04)
Age	-0.037 (0.01)***	-0.023 (0.00)***	-0.028 (0.00)***	-0.021 (0.00)***	-0.028 (0.00)***	-0.025
Age squared	0.00024 (0.04)*	0.000072 (0.00)	0.00023 (0.00)**	0.000046 (0.00)	0.00018 (0.00)*	0.000086 (0.00)
Total depressive symptoms*Age		-0.0080 (0.00)*				
Total depressive symptoms*Age squared		0.00026				
Negative attitude*Age			0.00075 (0.00)			-0.0020 (0.00)
Performance difficulties*Age				0.0011 (0.00)		-0.0011 (0.00)
Somatic complaints*Age					0.0039 (0.00)***	0.0042 (0.00)**
Random effects						
Variance of intercept	1.05 (0.05)*	0.53 (0.03)*	0.56 (0.03)*	0.56 (0.03)*	0.68 (0.03)*	0.70 (0.05)*
Variance of total depressive symptoms		0.21 (0.02)*				
Variance of negative attitude			0.19 (0.02)*			0.23 (0.03)*
Variance of performance difficulties				0.20 (0.02)*		0.27 (0.03)*
Variance of somatic complaints					0.20 (0.02)*	0.17 (0.02)*
Variance of age	0.028 (0.00)*	0.020 (0.00)*	0.021 (0.00)*	0.020 (0.00)*	0.024 (0.00)*	0.020 (0.00)*
Residual variance	0.62 (0.01)*	0.56 (0.01)*	0.57 (0.01)*	0.57 (0.01)*	0.59 (0.01)*	0.53 (0.01)*

*** p<.001 ** p<.01 * p<.05 *Note:* Depressive symptoms and paranoid ideation were measured in 1992, 1997, 2001, 2007, and 2012. All the models were adjusted for gender and socioeconomic factors. Model 1 was an unconditional model with age terms as predictors. In model 2, total score of depressive symptoms were added to the model. In models 3, depressive subsymptoms were added separately to the model. In model 4, depressive subsymptoms were added simultaneously to the model.

Table 3. Estimates and standard errors (SE) of fixed and random effects when predicting standardized scores of change in paranoid ideation by change in depressive symptoms (measured with the mBDI) and age.

	Model 5 (R^2=.63) Estimate (SE)
Fixed effects	
Intercept	-0.089 (0.08)
Change in depression	0.54 (0.07)***
Age	0.014 (0.01)
Age-squared	-0.00035 (0.00)
Change in depression*Age	-0.017 (0.01)*
Change in depression*Age-squared	0.00043 (0.00)*
Random effects	
Variance of intercept	0.00 (0.00)*
Residual variance	0.89 (0.01)*

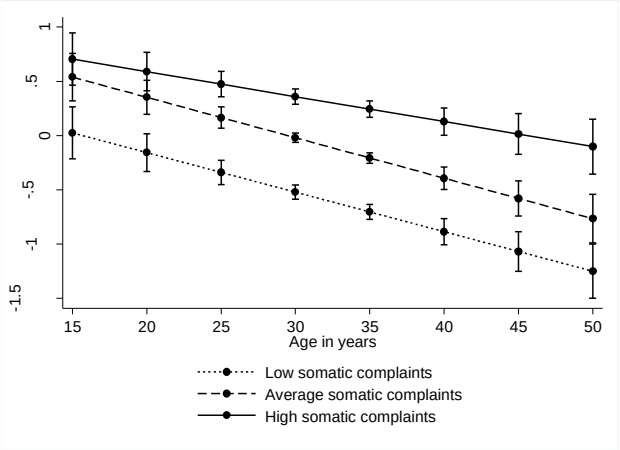
*** p<.001 ** p<.01 * p<.05
Note: The model was adjusted for gender, socioeconomic factors, baseline depressive symptoms, and baseline paranoid ideation in 1992.

Table 4. Regression coefficients (β) and standard errors (SE) of severity of depression (measured with the BDI-II) in 2007, 2011, and 2012, when predicting standardized scores of paranoid ideation in 2012.

	Depression in 2007 (n=1313, R^2= .52) β (SE)	Depression in 2011 (n=1077, R^2=.52) β (SE)	Depression in 2012 (n=1318, R^2=.54) β (SE)
Severity of depression[a]			
Mild	0.10 (0.08)	0.39 (0.09)***	0.40 (0.09)***
Moderate	0.06 (0.12)	0.13 (0.15)	0.38 (0.13)**
Severe	-0.07 (0.21)	1.07 (0.26)***	0.95 (0.18)***

Note: *** p<.001 ** p<.01 * p<.05 [a] Participants with no depression as the reference group. All the models were adjusted for age, gender, socioeconomic factors, and level of paranoid ideation in 2007.

a. Total depressive symptoms



b. Somatic complaints

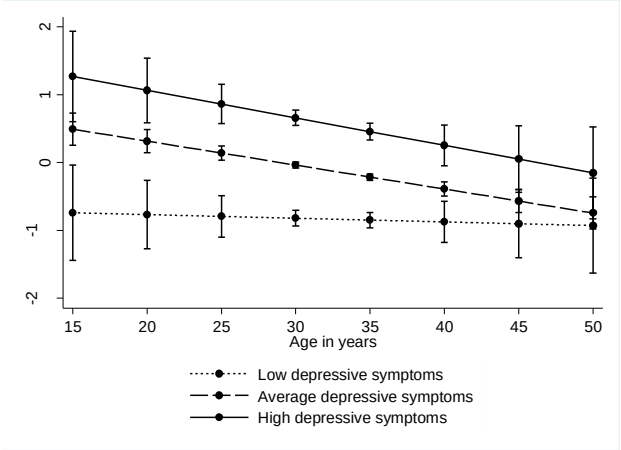


Figure 1. Growth trajectories of standardized scores of paranoid ideation separately for participants belonging to the lowest 10%, average, and the highest 10% in total depressive symptoms (a) and somatic complaints (b). Predicted means with 95% confidence intervals.

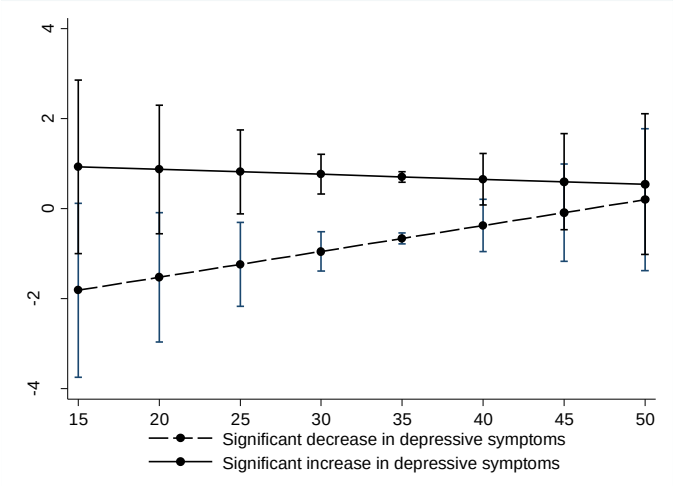


Figure 2. Growth trajectories for standardized scores of change in paranoid ideation separately for participants belonging to the largest 10% in increase and the largest 10% in decrease of depressive symptoms. Predicted means with 95% confidence intervals.

Supplemental Table 1. The numbers of observations for paranoid ideation and depressive symptoms in different measurement years.

	1992	1997	2001	2007	2011	2012	Total
Paranoid ideation	1617	1535	1656	1790	-	1510	8018
Depressive symptoms (the BDI-II)	-	-	-	1772	1580	1500	4852
Depressive symptoms (the mBDI)	1587	1515	1641	1776	-	1494	8012

Note: Paranoid ideation was measured in 1992, 1997, 2001, 2007, and 2012, depressive symptoms with the mBDI in 1992, 1997, 2001, 2007, and 2012, and depressive symptoms with the BDI-II in 2007, 2011, and 2012. "Total" refers to the total number of observations over all the measurement times.

Supplemental Table 2. The internal consistencies (Cronbach's alpha) for the scales of paranoid ideation and depressive symptoms in different measurement years.

	1992	1997	2001	2007	2011	2012
Paranoid ideation	.71	.74	.75	.78	-	.79
Total depressive symptoms (mBDI)	.87	.89	.92	.93	-	.93
Negative attitude (mBDI)	.75	.78	.81	.82	-	.83
Performance difficulties (mBDI)	.81	.85	.86	.88	-	.88
Somatic complaints (mBDI)	.59	.69	.71	.73	-	.73
Depressive symptoms (BDI-II)	-	-	-	.92	.91	.92

Note: Paranoid ideation was measured in 1992, 1997, 2001, 2007, and 2012, depressive symptoms with the mBDI in 1992, 1997, 2001, 2007, and 2012, and depressive symptoms with the BDI-II in 2007, 2011, and 2012.

Supplemental Table 3. The goodness-of-fit statistics for the longitudinal models on the predictive relationships between depressive symptoms and paranoid ideation.

							Model comparisons		
	χ^2 value	*df*	*p*	RMSEA	CFI	BIC	χ^2 difference test	*df*	*p*
Model 1	1628.940	24	<.001	.178	.813	38240.668			
Model 2	35.627	16	<.01	.024	.998	36708.588	χ^2(1 vs. 2)=1593.313	8	<.001
Model 3	24.269	12	<.05	.022	.999	36727.845	χ^2(2 vs. 3)=11.358	4	<.05

Note: Model 1: no cross-lagged predictive paths between depressive symptoms and paranoid ideation. Model 2: cross-lagged paths from depressive symptoms to paranoid ideation and vice versa (estimated to be equal in both directions). Model 3: cross-lagged paths freely estimated. RMSEA = the Root Mean Square Error of Approximation, CFI = the Comparative Fit Index, BIC = the Bayesian Information Criterion.